WEST Search History

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Updated Search 5/24/04

DATE: Monday, May 24, 2004

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		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=AND	
☐	L1	$methylase.clm.	210
☐	L2	$methyltransferase.clm.	272
☐	L3	$methyl-transferase.clm.	14
☐	L4	dam.clm.	3767
☐	L5	(L4 or l3 or l2 or l1)	4206
☐	L6	L5 and (mutant or mutagenesis or mutation or altered or alteration or modification or modified)	2832
☐	L7	L5 and (mutant or mutagenesis or mutation or altered or alteration or modification or modified).clm.	210
☐	L8	L7 and adenine.clm.	9
☐	L9	L7 and adeno$.clm.	10
☐	L10	L9 or l8	19
☐	L11	L5 and mahan	13
☐	L12	L5 and mahan.in.	7
☐	L13	('6596701' |'6610504' |'6020139' |'5856095' |'6642434')!.PN.	10

END OF SEARCH HISTORY

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☐ 1. 20040014083. 24 Feb 03. 22 Jan 04. Detection of heteroduplex polynucleotides using mutant nucleic acid repair enzymes with attenuated catalytic activity. Yuan, Chong-Sheng, et al. 435/6; C12Q001/68.

☐ 2. 20030039073. 01 Apr 02. 27 Feb 03. Disc head slider designs to reduce particle sensitivity and improve disc following capability. Rao, Ram Mahan. 360/235.8; 360/236.2 G11B005/60.

☐ 3. 20020086332. 09 Aug 01. 04 Jul 02. Method of reducing bacterial proliferation. Mahan, Michael J., et al. 435/7.1; G01N033/53.

☐ 4. 20020086032. 09 Aug 01. 04 Jul 02. Producing antibodies with attenuated bacteria with altered DNA adenine methylase activity. Mahan, Michael J., et al. 424/200.1; 435/252.3 A61K039/02 C12N001/21.

☐ 5. 20020081317. 09 Aug 01. 27 Jun 02. Bacteria with altered DNA adenine methylase (DAM) activity and heterologous epitope. Mahan, Michael J., et al. 424/200.1; 435/252.3 435/320.1 A61K039/02 C12N001/21 C12N015/74.

☐ 6. 20020077272. 09 Aug 01. 20 Jun 02. Reducing bacterial virulence. Mahan, Michael J., et al. 514/1; 514/263.4 A61K031/00 A61K031/52.

☐ 7. 20020076417. 09 Aug 01. 20 Jun 02. Attenuated bacteria with altered DNA adenine methylase activity. Mahan, Michael J., et al. 424/200.1; 435/252.3 435/252.33 435/252.35 A61K039/02 C12N001/21.

☐ 8. 20020068068. 09 Aug 01. 06 Jun 02. Method of creating antibodies and compositions used for same. Mahan, Michael J., et al. 424/200.1; 424/257.1 424/258.1 424/261.1 A61K039/108 A61K039/112 A61K039/106 A61K039/02.

☐ 9. 6610504. 10 Apr 00; 26 Aug 03. Methods of determining SAM-dependent methyltransferase activity using a mutant SAH hydrolase. Yuan; Chong-Sheng. 435/15; 435/18. C12Q001/48 C12Q001/34.

☐ 10. 6221849. 24 Jun 98; 24 Apr 01. DNA methyltransferase genomic sequences and antisense oligonucleotides. Szyf; Moshe, et al. 514/44; 435/375 536/24.5. A61K031/7088 A61K031/712 A61K031/7125 C07H021/04.

☐ 11. 6184211. 30 May 96; 06 Feb 01. Inhibition of DNA methyltransferase. Szyf; Moshe. 514/44; 424/130.1 435/183 435/6 435/7.1 536/24.5. A61K048/00 A61K039/395 C07H021/04 C12Q001/68.

☐ 12. 6020318. 30 May 97; 01 Feb 00. DNA methyltransferase genomic sequences and antisense oligonucleotides. Szyf; Moshe, et al. 514/44; 536/24.5. A61K031/70 C07H021/00.

☐ 13. 5919772. 07 Jun 95; 06 Jul 99. Antisense oligonucleotides having tumorigenicity-inhibiting activity. Szyf; Moshe, et al. 514/44; 536/24.5. A61K048/00 C07H021/04.

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Search Results - Record(s) 1 through 10 of 10 returned.

L13: Entry 1 of 10 File: USPT Nov 4, 2003

US-PAT-NO: 6642434
DOCUMENT-IDENTIFIER: US 6642434 B1

TITLE: Transgenic plants with .gamma.-tocopherol methyltransferase

DATE-ISSUED: November 4, 2003

US-CL-CURRENT: 800/278; 435/410, 435/419, 435/69.1, 435/70.1, 536/23.1, 536/23.2, 536/23.6, 800/295, 800/298, 800/306

INT-CL: [07] C12 N 15/29, C12 N 5/04, C12 N 15/82, C12 P 21/06, C07 H 21/04

L13: Entry 2 of 10 File: USPT Aug 26, 2003

US-PAT-NO: 6610504
DOCUMENT-IDENTIFIER: US 6610504 B1

TITLE: Methods of determining SAM-dependent methyltransferase activity using a mutant SAH hydrolase

DATE-ISSUED: August 26, 2003

US-CL-CURRENT: 435/15; 435/18

INT-CL: [07] C12 Q 1/48, C12 Q 1/34

L13: Entry 3 of 10 File: USPT Jul 22, 2003

US-PAT-NO: 6596701
DOCUMENT-IDENTIFIER: US 6596701 B1

TITLE: S-adenosyl methionine regulation of metabolic pathways and its use in diagnosis and therapy

DATE-ISSUED: July 22, 2003

US-CL-CURRENT: 514/46; 435/7.1, 528/338, 528/340

INT-CL: [07] A01 N 43/04, G01 N 33/53, C08 G 69/26

L13: Entry 4 of 10 File: USPT Feb 1, 2000

US-PAT-NO: 6020139
DOCUMENT-IDENTIFIER: US 6020139 A

TITLE: S-adenosyl methionine regulation of metabolic pathways and its use in diagnosis and therapy

DATE-ISSUED: February 1, 2000

US-CL-CURRENT: 435/7.1; 435/192, 514/556

INT-CL: [06] G01 N 33/53, C12 N 9/08, A01 N 37/30

L13: Entry 5 of 10 File: USPT Jan 5, 1999

US-PAT-NO: 5856095
DOCUMENT-IDENTIFIER: US 5856095 A
**** See image for Certificate of Correction ****

TITLE: Identification of two novel mutant alleles of human thiopurine S-methyltransferase, and diagnostic uses thereof

DATE-ISSUED: January 5, 1999

US-CL-CURRENT: 435/6; 435/810, 435/91.2, 536/23.5, 536/24.31, 536/24.33

INT-CL: [06] C12 Q 1/68, C12 P 19/34, C07 H 21/04

L13: Entry 6 of 10 File: DWPI Aug 26, 2003

DERWENT-ACC-NO: 2003-842412
ABSTRACTED-PUB-NO: US 6610504B
COPYRIGHT 2004 DERWENT INFORMATION LTD

TITLE: Assaying S-adenosylmethionine (SAM)-dependent methyltransferase comprises converting SAM to S-adenosylhomocysteine (SAH), contacting with mutant SAH hydrolase and detecting SAH-mutant hydrolase and SAH binding

INT-CL (IPC): C12 Q 1/34, C12 Q 1/48
Derwent-CL (DC): B04, D16
CPI Codes: B04-L04; B04-L05; B11-C07B3; B11-C08E3; B11-C08E6; B12-K04A; B12-K04E; D05-A02B; D05-H09;

L13: Entry 7 of 10 File: DWPI Feb 4, 1999

DERWENT-ACC-NO: 1999-142458
ABSTRACTED-PUB-NO: WO 9904622A
COPYRIGHT 2004 DERWENT INFORMATION LTD

TITLE: Newly isolated DNA fragment comprising a ~c-tocopherol (vitamin E) methyltransferase coding sequence - useful for producing ~a-tocopherol, and transgenic plants, seeds and oils with an altered tocopherol profile

INT-CL (IPC): A01 H 5/00, A01 H 5/10, C07 H 21/04, C12 N 5/04, C12 N 9/10, C12 N 15/29, C12 N 15/63, C12 N 15/82, C12 P 21/06
Derwent-CL (DC): C06, D16 , P13
CPI Codes: C03-H; C04-A0800E; C04-A09F; C04-B01C; C04-E02E; D05-C10; D05-H12A; D05-H12E; D05-H16B; D05-H17A3;

L13: Entry 8 of 10 File: DWPI Jan 5, 1999

DERWENT-ACC-NO: 1999-105094

ABSTRACTED-PUB-NO: US 5856095A
COPYRIGHT 2004 DERWENT INFORMATION LTD

TITLE: Mutant alleles of thiopurine S-methyl-transferase gene - used to detect mutations associated with TPMT deficiency, which can cause potentially fatal toxicity in cancer patients treated with e.g. mercaptopurine

INT-CL (IPC): C07 H 21/04, C12 P 19/34, C12 Q 1/68
Derwent-CL (DC): B04, D16
CPI Codes: B04-E02E; B04-E05; B04-L04; B11-C08E4; B11-C08E5; B12-K04A; B12-K04F; D05-H09; D05-H12B; D05-H12D; D05-H18B;

L13: Entry 9 of 10 File: DWPI Jul 22, 2003

DERWENT-ACC-NO: 1996-497351
ABSTRACTED-PUB-NO: US 6020139A
COPYRIGHT 2004 DERWENT INFORMATION LTD

TITLE: Modulation of S-adenosyl-L-methionine metabolism - used for diagnosis and treatment of conditions such as cancer, multiple sclerosis, diabetes and arthritis

INT-CL (IPC): A01 N 37/30, A01 N 43/04, A61 K 31/00, A61 K 31/195, A61 K 31/415, A61 K 31/455, A61 K 31/70, A61 K 33/00 , A61 K 38/00, A61 K 39/00, A61 K 45/00, A61 K 45/05, A61 K 49/00, A61 K 51/00, A61 P 43/00, C07 C 321/00 , C07 C 323/00, C07 C 381/00, C07 D 211/72, C07 D 211/84, C07 D 231/56, C07 D 235/00, C07 D 235/01, C07 H 19/00, C07 H 19/207, C07 H 21/00, C08 G 69/26, C12 N 9/00, C12 N 9/08, C12 P 1/00, C12 Q 1/00, C12 Q 1/68, C12 Q 1/70, G01 N 31/00, G01 N 33/00, G01 N 33/15, G01 N 33/48, G01 N 33/50, G01 N 33/53, G01 N 33/532, G01 N 33/535, G01 N 33/536, G01 N 33/543, G01 N 33/557, G01 N 33/558, G01 N 33/564, G01 N 33/566, G01 N 33/72, G01 N 33/86, G01 N 33/92
Derwent-CL (DC): B04, D16 , J04 , S03
CPI Codes: B11-C08E; B12-K04A; B14-C09; B14-H01; B14-N17B; B14-S01; B14-S04; D05-A02; D05-H09; J04-B01;
EPI Codes: S03-E09; S03-E14; S03-E14H; S03-E14H4;

L13: Entry 10 of 10 File: DWPI

DERWENT-ACC-NO: 1968-77425P
ABSTRACTED-PUB-NO: NL 6610504A
COPYRIGHT 2004 DERWENT INFORMATION LTD

TITLE: Stable concentrated aqueous solution of urea formaldehyde and their re - action products

INT-CL (IPC): C08G 9/10
Derwent-CL (DC): A22, A81 , G03
CPI Codes: A05-B03; A10-E; A12-A;

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Search Results - Record(s) 1 through 7 of 7 returned.

☐ 1. Document ID: US 20030039073 A1

Using default format because multiple data bases are involved.

L12: Entry 1 of 7 File: PGPB Feb 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030039073
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030039073 A1

TITLE: Disc head slider designs to reduce particle sensitivity and improve disc following capability

PUBLICATION-DATE: February 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Rao, Ram Mahan	Roseville	MN	US	

US-CL-CURRENT: 360/235.8; 360/236.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

☐ 2. Document ID: US 20020086332 A1

L12: Entry 2 of 7 File: PGPB Jul 4, 2002

DOCUMENT-IDENTIFIER: US 20020086332 A1
TITLE: Method of reducing bacterial proliferation

INVENTOR:
Mahan, Michael J.

CLAIMS:

1. A method of reducing bacterial virulence, comprising: contacting bacteria with an agent that alters the bacteria's native level of DNA methyltransferase (Dam) activity thereby altering the bacteria's native level of methylation of adenine in a GATC tetranucleotide of the bacteria, and thereby inhibiting virulence of the bacteria.

2. The method of claim 1, wherein the agent reduces the bacteria's native level of

DNA methyltransferase activity.

3. The method of claim 1, wherein the agent reduces the Dam activity by reducing the bacteria's level of expression of Dam.

4. The method of claim 1, wherein the agent reduces the Dam activity by blocking a Dam interaction site.

5. The method of claim 1, wherein the agent increases the bacteria's native level of DNA methyltransferase activity.

6. The method of claim 1, wherein the agent reduces the bacteria's native level of methylated adenine in a GATC tetranucleotide by inhibiting DNA methyltransferase activity.

7. The method of claim 1, wherein the agent increases the bacteria's native level of methylated adenine in a GATC tetranucleotide by increasing DNA methyltransferase activity.

8. The method of claim 1, wherein the agent binds a Dam enzyme.

9. The method of claim 1, wherein the agent binds a native sequence of a bacteria and decreases expression of Dam below a normal level.

10. The method of claim 1, wherein the agent binds a native sequence of a bacteria and increases expression of Dam above a normal level.

11. The method of claim 1, wherein the agent alters Dam activity of a pathogenic bacteria selected from the group consisting of Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus somnus, Actinobacillus pleuropneumoniae, Pasteurella multocida, Mannheimia haemolytica, NT Haemophilus influenzae, Helicobacter pyiori and Shigella spp.

12. The method of claim 1, wherein the agent alters native Dam activity of a pathogenic bacteria selected from the group consisting of Escherichia, Vibrio, Yersinia and Salmonella.

19. A method of reducing pathogenicity of a pathogenic bacteria, comprising: administering an agent that alters a pathogenic bacteria's native DNA adenine methylase (Dam) activity thereby altering the bacteria's native DNA methylation activity to an extent that the bacteria's pathogenicity is reduced.

20. The method of claim 19, wherein the agent reduces the Dam activity by reducing the bacteria's level of expression of Dam.

21. The method of claim 19, wherein the agent reduces the Dam activity by blocking a Dam interaction site.

22. The method of claim 19, wherein the agent increases Dam activity.

23. The method of claim 19, wherein the agent decreases Dam activity.

24. A method of treating a bacterial infection, comprising the steps of: administering to a subject infected with a pathogenic bacteria a therapeutically effective amount of a composition comprising a pharmaceutically acceptable carrier and an active agent that alters the bacteria's native level of DNA methyltransferase (Dam) activity; and allowing the agent to contact the bacteria for for a period of time and under conditions so as to inhibit proliferation of the bacteria.

25. The method of claim 24, wherein the agent reduces the Dam activity by reducing the bacteria's level of expression of Dam.

26. The method of claim 24, wherein the agent reduces the Dam activity by blocking a Dam interaction site.

27. The method of claim 24, wherein the agent reduces the level of Dam activity thereby reducing methylation of adenine in a GATC tetranucleotide in the bacteria, thereby inhibiting virulence of the bacteria.

28. The method of claim 24, wherein the agent increases the level of Dam activity thereby increasing methylation of adenine in a GATC tetranucleotide in the bacteria, thereby inhibiting virulence of the bacteria.

32. A method for treating bacterial infection comprising administering an agent that reduces the level or activity of a DNA methyltransferase thereby reducing methylation of adenine in a GATC tetranucleotide in the bacteria, thereby inhibiting the virulence of the bacteria.

33. The method of claim 32, wherein the reduction of the level of methylated adenine in a GATC tetranucleotide is effected by inhibiting DNA methyltransferase activity.

34. A composition for controlling bacterial pathogenicity, comprising: a carrier; and a compound that alters native DNA adenine methylase (Dam) activity.

36. The composition of claim 34, wherein the agent binds a Dam enzyme.

37. The composition of claim 34, wherein the agent which binds a native sequence of a bacteria and decreases expression of Dam below a normal level.

38. The composition of claim 34, wherein the agent which binds a native sequence of a bacteria and increases expression of Dam above a normal level.

40. The composition of claim 34, wherein the agent alters native Dam activity of a pathogenic bacteria selected from the group consisting of Escherichia, Vibrio, Yersinia and Salmonella.

☐ 3. Document ID: US 20020086032 A1

L12: Entry 3 of 7 File: PGPB Jul 4, 2002

DOCUMENT-IDENTIFIER: US 20020086032 A1
TITLE: Producing antibodies with attenuated bacteria with altered DNA adenine methylase activity

INVENTOR:
Mahan, Michael J.

CLAIMS:

1. A method, comprising the steps of: administering to a subject capable of generating an immune response a composition comprising a pharmaceutically acceptable acceptable excipient an immunogenic dose of attenuated bacteria which bacteria comprise altered DNA adenine methylase (Dam) activity relative to a wild-type bacteria; and allowing the composition to remain in the subject for a time and under conditions to allow the subject to generate an immune response to the attenuated bacteria and produce antibodies specific to the attenuated bacteria.

32. A method of eliciting an immune response in an individual, comprising: administering an immunogenic composition to an individual in an amount sufficient to elicit an immune response wherein the composition comprises a pharmaceutically acceptable carrier and a bacteria comprising a genome characterized by a mutation altering DNA adenine methylase (Dam) activity such that the bacteria is attenuated; allowing the composition to remain in the individual for a time and under conditions to allow the individual to generate an immune response.

34. An immunogenic composition, comprising: a pharmaceutically acceptable excipient; and live bacteria, said bacteria comprising altered DNA adenine methylase (Dam) activity wherein the altered activity reduces virulence relative to the bacteria with wild-type Dam activity.

35. The immunogenic composition of claim 34, wherein the Dam activity is altered by a heterologous nucleotide.

36. The immunogenic composition of claim 34, wherein the Dam activity is altered by a mutation in the bacteria's genome which mutation alters a gene involved in expressing Dam in a manner selected from the group consisting of reduced expression, expression, no expression, overexpression, expression of a form of Dam altered from Dam native to the bacteria.

37. An attenuated strain of a bacteria, said bacteria comprising altered DNA adenine methylase (Dam) activity such that the bacteria are attenuated.

38. The attenuated strain of claim 37, wherein the altered activity reduces Dam activity.

39. The attenuated strain of claim 37, wherein the altered activity eliminates Dam activity.

40. The attenuated strain of claim 37, wherein the altered activity is obtained by a deletion in a dam gene.

41. The attenuated strain of claim 37, wherein the altered activity is obtained by an increase in expression of Dam.

☐ 4. Document ID: US 20020081317 A1

L12: Entry 4 of 7 File: PGPB Jun 27, 2002

DOCUMENT-IDENTIFIER: US 20020081317 A1
TITLE: Bacteria with altered DNA adenine methylase (DAM) activity and heterologous epitope

INVENTOR:
Mahan, Michael J.

CLAIMS:

1. An immunogenic composition, comprising: a pharmaceutically acceptable excipient; and live bacteria with DNA adenine methylase (Dam) activity altered relative to wild-type activity of an unaltered pathogenic bacteria, with the alteration being in a manner which renders the bacteria attenuated; and a first heterologous nucleotide sequence operatively inserted in the bacteria which first heterologous sequence expresses a heterologous antigen.

2. The immunogenic composition of claim 1, wherein the Dam activity is altered by an artificially engineered change in the pathogenic bacteria's genome.

3. The immunogenic composition of claim 1, wherein the Dam activity is altered by a second heterologous nucleotide sequence.

☐ 5. Document ID: US 20020077272 A1

L12: Entry 5 of 7 File: PGPB Jun 20, 2002

DOCUMENT-IDENTIFIER: US 20020077272 A1
TITLE: Reducing bacterial virulence

INVENTOR:
Mahan, Michael J.

CLAIMS:

1. A method of reducing bacterial virulence, comprising: contacting bacteria with an agent that alters the bacteria's native level of DNA methyltransferase (Dam) activity thereby altering the bacteria's native level of methylation of adenine in a GATC tetranucleotide of the bacteria, and thereby inhibiting virulence of the bacteria.

6. The method of claim 1, wherein the agent reduces the bacteria's native level of DNA methyltransferase activity.

7. The method of claim 1, wherein the agent reduces the Dam activity by reducing the bacteria's level of expression of Dam.

8. The method of claim 1, wherein the agent reduces the Dam activity by blocking a Dam interaction site.

9. The method of claim 1, wherein the agent increases the bacteria's native level of DNA methyltransferase activity.

10. The method of claim 1, wherein the agent reduces the bacteria's native level of

methylated adenine in a GATC tetranucleotide by inhibiting DNA methyltransferase activity.

11. The method of claim 1, wherein the agent increases the bacteria's native level of methylated adenine in a GATC tetranucleotide by increasing DNA methyltransferase activity.

12. The method of claim 1, wherein the agent binds a Dam enzyme.

13. The method of claim 1, wherein the agent binds a native sequence of a bacteria and decreases expression of Dam below a normal level.

14. The method of claim 1, wherein the agent binds a native sequence of a bacteria and increases expression of Dam above a normal level.

15. The method of claim 1, wherein the agent alters Dam activity of a pathogenic bacteria selected from the group consisting of Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus somnus, Actinobacillus pleuropneumoniae, Pasteurella multocida, Mannheimia haemolytica, NT Haemophilus influenzae, Helicobacter pyiori and Shigella spp.

16. The method of claim 1, wherein the agent alters native Dam activity of a pathogenic bacteria selected from the group consisting of Escherichia, Vibrio, Yersinia and Salmonella.

19. A method of reducing pathogenicity of a pathogenic bacteria, comprising: administering an agent that alters a pathogenic bacteria's native DNA adenine methylase (Dam) activity thereby altering the bacteria's native DNA methylation activity to an extent that the bacteria's pathogenicity is reduced.

20. The method of claim 19, wherein the agent reduces the Dam activity by reducing the bacteria's level of expression of Dam.

21. The method of claim 19, wherein the agent reduces the Dam activity by blocking a Dam interaction site.

22. The method of claim 19, wherein the agent increases Dam activity.

23. The method of claim 19, wherein the agent decreases Dam activity.

24. A method of treating a bacterial infection, comprising the steps of: administering to a subject infected with a pathogenic bacteria a therapeutically effective amount of a composition comprising a pharmaceutically acceptable carrier and an active agent that alters the bacteria's native level of DNA methyltransferase (Dam) activity; and allowing the agent to contact the bacteria for for a period of time and under conditions so as to inhibit proliferation of the bacteria.

25. The method of claim 24, wherein the agent reduces the Dam activity by reducing the bacteria's level of expression of Dam.

26. The method of claim 24, wherein the agent reduces the Dam activity by blocking a Dam interaction site.

27. The method of claim 24, wherein the agent reduces the level of Dam activity thereby reducing methylation of adenine in a GATC tetranucleotide in the bacteria, thereby inhibiting virulence of the bacteria.

28. The method of claim 24, wherein the agent increases the level of Dam activity thereby increasing methylation of adenine in a GATC tetranucleotide in the

bacteria, thereby inhibiting virulence of the bacteria.

32. A method for treating bacterial infection comprising administering an agent that that reduces the level or activity of a DNA methyltransferase thereby reducing methylation of adenine in a GATC tetranucleotide in the bacteria, thereby inhibiting the virulence of the bacteria.

33. The method of claim 32, wherein the reduction of the level of methylated adenine in a GATC tetranucleotide is effected by inhibiting DNA methyltransferase activity.

34. A composition for controlling bacterial pathogenicity, comprising: a carrier; and a compound that alters native DNA adenine methylase (Dam) activity.

36. The composition of claim 34, wherein the agent binds a Dam enzyme.

37. The composition of claim 34, wherein the agent which binds a native sequence of a bacteria and decreases expression of Dam below a normal level.

38. The composition of claim 34, wherein the agent which binds a native sequence of a bacteria and increases expression of Dam above a normal level.

39. An attenuated strain of a bacteria, said bacteria comprising altered DNA adenine methylase (Dam) activity such that the bacteria are attenuated.

40. The attenuated strain of claim 1, wherein the altered activity reduces Dam activity.

41. The attenuated strain of claim 1, wherein the altered activity eliminates Dam activity.

42. The attenuated strain of claim 1, wherein the altered activity is obtained by a deletion in a dam gene.

43. The attenuated strain of claim 1, wherein the altered activity is obtained by an increase in expression of Dam.

☐ 6. Document ID: US 20020076417 A1

L12: Entry 6 of 7 File: PGPB Jun 20, 2002

DOCUMENT-IDENTIFIER: US 20020076417 A1
TITLE: Attenuated bacteria with altered DNA adenine methylase activity

INVENTOR:
Mahan, Michael J.

CLAIMS:

1. An attenuated strain of a bacteria, said bacteria comprising altered DNA adenine methylase (Dam) activity such that the bacteria are attenuated.

2. The attenuated strain of claim 1, wherein the altered activity reduces Dam activity.

3. The attenuated strain of claim 1, wherein the altered activity eliminates Dam activity.

4. The attenuated strain of claim 1, wherein the altered activity is obtained by a deletion in a dam gene.

5. The attenuated strain of claim 1, wherein the altered activity is obtained by an increase in expression of Dam.

12. The attenuated strain of claim 11, wherein the heterologous nucleotide is operatively inserted into a plasmid and expresses DNA adenine methylase.

21. A composition, comprising: a pharmaceutically acceptable excipient; and bacteria with altered DNA adenine methylase (Dam) activity which altered DNA adenine adenine methylase activity renders the bacteria non-pathogenic.

23. An immunogenic composition, comprising: a pharmaceutically acceptable excipient; and live bacteria, said bacteria comprising altered DNA adenine methylase (Dam) activity wherein the altered activity reduces virulence relative to the bacteria with wild-type Dam activity.

24. The immunogenic composition of claim 23, wherein the Dam activity is altered by a heterologous nucleotide.

25. The immunogenic composition of claim 23, wherein the Dam activity is altered by a mutation in the bacteria's genome which mutation alters a gene involved in expressing Dam in a manner selected from the group consisting of reduced expression, expression, no expression, overexpression, expression of a form of Dam altered from Dam native to the bacteria.

26. A method, comprising the steps of: administering to a subject capable of generating an immune response a composition comprising a pharmaceutically acceptable excipient an immunogenic dose of altered bacteria with altered DNA adenine methylase (Dam) activity which bacteria are attenuated; and allowing the composition to remain in the subject for a time and under conditions to allow the subject to generate an immune response to the bacteria and produce antibodies specific to the bacteria.

33. A method of eliciting an immune response in an individual, comprising: administering an immunogenic composition to an individual in an amount sufficient to elicit an immune response wherein the composition comprises a pharmaceutically acceptable carrier and a bacteria comprising a genome characterized by a mutation altering DNA adenine methylase (Dam) activity such that the bacteria is attenuated; allowing the composition to remain in the individual for a time and under conditions to allow the individual to generate an immune response.

☐ 7. Document ID: US 20020068068 A1

L12: Entry 7 of 7 File: PGPB Jun 6, 2002

DOCUMENT-IDENTIFIER: US 20020068068 A1
TITLE: Method of creating antibodies and compositions used for same

INVENTOR:
Mahan, Michael J.

CLAIMS:

1. A composition, comprising: a pharmaceutically acceptable excipient; and bacteria with altered DNA adenine methylase activity which altered DNA adenine methylase activity renders the bacteria non-pathogenic.

5. The composition of claim 4, wherein the heterologous nucleotide is operatively inserted into a plasmid and expresses DNA adenine methylase.

15. An immunogenic composition, comprising: a pharmaceutically acceptable excipient; and live bacteria, said bacteria comprising altered DNA adenine methylase (Dam) activity wherein the altered activity reduces virulence relative to the bacteria with wild-type Dam activity.

16. The immunogenic composition of claim 15, wherein the Dam activity is altered by a heterologous nucleotide.

17. The immunogenic composition of claim 15, wherein the Dam activity is altered by a mutation in the bacteria's genome which mutation alters a gene involved in expressing Dam in a manner selected from the group consisting of reduced expression, expression, no expression, overexpression, expression of a form of Dam altered from Dam native to the bacteria.

18. An attenuated strain of a pathogenic bacteria, said bacteria containing a mutation which alters Dam activity such that the bacteria are attenuated.

19. The attenuated strain of claim 18, wherein the mutation reduces Dam activity.

20. The attenuated strain of claim 18, wherein the mutation eliminates Dam activity.

21. The attenuated strain of claim 18, wherein the mutation is a deletion in a dam gene.

22. The attenuated strain of claim 18, wherein the mutation causes an increase in expression of Dam.

26. A method, comprising the steps of: administering to a subject capable of generating an immune response a composition comprising a pharmaceutically acceptable excipient an immunogenic dose of altered bacteria with altered DNA adenine methylase (Dam) activity which bacteria are attenuated; and allowing the composition to remain in the subject for a time and under conditions to allow the subject to generate an immune response to the bacteria and produce antibodies specific to the bacteria.

33. A method of eliciting an immune response in an individual, comprising: administering an immunogenic composition to an individual in an amount sufficient to elicit an immune response wherein the composition comprises a pharmaceutically acceptable carrier and a bacteria comprising a genome characterized by a mutation altering DNA adenine methylase (Dam) activity such that the bacteria is attenuated; allowing the composition to remain in the individual for a time and under conditions to allow the individual to generate an immune response.

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. Document ID: US 6642434 B1

L10: Entry 15 of 19 File: USPT Nov 4, 2003

DOCUMENT-IDENTIFIER: US 6642434 B1
TITLE: Transgenic plants with .gamma.-tocopherol methyltransferase

CLAIMS:

2. An isolated DNA construct comprising an Arabidopsis .gamma.-tocopherol methyltransferase coding sequence operably connected to a plant promoter not natively associated with the coding seguence.

3. A genetic construct comprising a .gamma.-tocopherol methyltransferase coding sequence operably connected to a plant promoter not natively associated with the coding sequence, wherein the .gamma.-tocopherol methyltransferase coding sequence is selected from the group consisting of SEQ ID NO:1 and SEQ ID NO:3.

5. The plant of claim 4, wherein the plant has an altered .alpha.-tocopherol:.gamma.-tocopherol ratio relative to an untransformed wild-type plant.

7. The plant of claim 4, wherein the plant has an altered .delta.-tocopherol:.beta.-tocopherol ratio relative to an untransformed wild-type plant.

9. A transgenic plant having an altered relative proportion of tocopherols in its tissues as compared to non-transgenic plants of the same species, the transgenic plant comprising in its genome an inserted .gamma.-tocopherol methyltransferase coding sequence, the .gamma.-tocopherol methyltransferase coding sequence selected from the group consisting of SEQ ID NO:1 and SEQ ID NO:3.

10. The plant of claim 9 wherein the .gamma.-tocopherol methyltransferase is in the sense orientation.

11. The plant of claim 9 wherein the .gamma.-tocopherol methyltransferase is in its antisense orientation.

12. A method of producing .alpha.-tocopherol comprising the steps of: (a) constructing an expression host cell comprising in its genome a .gamma.-tocopherol methyltransferase coding sequence operably connected to a promoter not natively associated with the sequence, wherein the promoter is functional in the host cell, the .gamma.-tocopherol methyltransferase gene encoding proteins selected from the group consisting of SEQ ID NO:2 and SEQ ID NO:4; (b) culturing the host cell under conditions suitable to allow expression of the .gamma.-tocopherol methyltransferase; and (c) reacting .gamma.-tocopherol and S-adenosylmethionine with with the .gamma.-tocopherol methyltransferase protein of step b under suitable conditions and for a period of time sufficient to allow conversion of .gamma.-tocopherol to .alpha.-tocopherol.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

☐ 16. Document ID: US 6610504 B1

L10: Entry 16 of 19 File: USPT Aug 26, 2003

DOCUMENT-IDENTIFIER: US 6610504 B1
TITLE: Methods of determining SAM-dependent methyltransferase activity using a mutant SAH hydrolase

CLAIMS:

1. A method for assaying for the activity of a S-adenosylmethionine (SAM)-dependent methyltransferase, comprising: a) contacting a SAM-dependent methyltransferase with a substrate of the methyltransferase in the presence of SAM, whereby a methyl group is transferred from the methyltransferase to the substrate and the SAM is converted into S-adenosylhomocysteine (SAH); b) contacting the resulting SAH with a mutant SAH SAH hydrolase which substantially retains its binding affinity or has enhanced binding affinity for SAH but has attenuated catalytic activity; and c) detecting binding between the SAH and the mutant SAH hydrolase to detect or determine the presence or amount of the SAH, whereby the activity of the SAM-dependent methyltransferase is assessed.

2. The method of claim 1, wherein the SAM-dependent methyltransferase is selected from the group consisting of a protein methyltransferase, a nucleic acid methyltransferase, a lipid methyltransferase, a polysaccharide methyltransferase and and a small molecule methyltransferase.

3. The method of claim 2, wherein the nucleic acid methyltransferase is a DNA methyltransferase or a RNA methyltransferase.

4. The method of claim 1, wherein the SAM-dependant methyltransferase comprises an amino acid consensus sequence selected from the group consisting of motif I ((V/I/L)(L/V)(D/E)(V/I)G(G/C)G(T/P)G), motif II ((P/G)(Q/T)(F/Y/A)DA(I/V/Y)(F/I)(C/V/L)) and motif III (LL(R/K)PGG(R/I/L)(L/I)(L/F/I/V)(I/L) of combinations thereof.

5. The method of claim 4, wherein the SAM-dependent methyltransferase comprises all the motifs I, II and III in the order of N'-I-II-III-C', the distance between the last amino acid residue of motif I and the first amino acid residue of motif II is from about 36 to about 90 amino acid residues, and the distance between the last amino acid residue of motif II and the first amino acid residue of motif III is from about 12 to about 38 amino acid residues.

6. The method of claim 4, wherein the SAM-dependent methyltransferase comprises the motif I only or comprises the motifs I and III only.

7. The method of claim 1, wherein the method for assaying for the activity of the methyltransferase is a diagnostic assay.

8. The method of claim 1, wherein the method for assaying for the activity of the methyltransferase is an assay for screening for compounds that modulate the activity activity of the methyltransferase.

9. The method of claim 1, further comprising comparing the activity of the methyltransferase to a control, whereby a change in the activity is detected.

10. The method of claim 1, wherein the mutant SAH hydrolase has attenuated hydrolytic activity but substantially retains its oxidative activity when compared to a wildtype SAH hydrolase from which the mutant SAH hydrolase is derived.

11. The method of claim 1, wherein the SAH is contacted with the mutant SAH hydrolase in the presence of a labeled SAH or a derivative or an analog thereof, whereby the amount of the labeled SAH bound to the mutant SAH hydrolase inversely

relates to amount of the SAH produced in step a).

13. The method of claim 1, wherein the mutant SAH hydrolase is a labeled mutant SAH hydrolase.

15. The method of claim 1, wherein the mutant SAH hydrolase is linked to a solid support.

17. The method of claim 16, wherein the mutant SAH hydrolases are arranged in an array on the solid support.

☐ 17. Document ID: US 6596701 B1

L10: Entry 17 of 19 File: USPT Jul 22, 2003

DOCUMENT-IDENTIFIER: US 6596701 B1
TITLE: S-adenosyl methionine regulation of metabolic pathways and its use in diagnosis and therapy

CLAIMS:

8. The method of claim 7 wherein said identifying abnormalities step of (b) comprises determining in said biological sample: (1) the concentration of metabolites selected from the group consisting of: (A) for Pathway 1, S-adenosylmethionine (SAM), S-adenosyl homocysteine (SAH), adenosine, homocysteine, glutathione, glutathione disulfide, cystathionine, .alpha.-ketobutyrate, cysteine, cystine, taurine, choline, betaine, dimethylglycine, methylglycine, glycine, serine, folate, tetrahydrofolate, methylene tetrahydrofolate, methyltetrahydrofolate, adenosine triphosphate, methylated vitamin B12, an O- or N-methylated product of a methylating enzyme and a demethylated product of a demethylating enzyme. (B) for Pathway 2, SAM, decarboxylated adenosylmethionine, methylthioadenosine, ornithine, putrescine, spermine, spermidine, N.sup.1 -acetylspermidine, and N.sup.1 -acetylspermine; and (C) for Pathway 8, biotin, biocytin, and biotin-protein conjugates, (2) the concentration or activity of enzymes, or the level of cellular functions, selected from the group consisting of: (A) for Pathway 1, adenosylmethionine synthetase, N.sup.5 -methyltetrahydrofolate:homocysteine methyltransferase, betaine:homocysteine methyltransferase, cystathionine .beta.-synthase, .gamma.-cystathionase, methylene tetrahydrofolate reductase, SAH hydrolase, an O-methyltransferase, an N-methyltransferase, adenosine deaminase, a glutathione synthetic enzyme and a glutathione degradative enzyme; (B) for Pathway 2, adenosinemethionine decarboxylase, spermidine synthetase, spermine synthetase, spermidine-spermine N.sup.1 -acetyltransferase, polyamine oxidase, methylthioadenosine phosphorylase, ornithine decarboxylase and polyamine transport; and (C) for Pathway 8, biotinase, pyruvate carboxylase, acetyl-CoA carboxylase, propionyl-CoA carboxylase, 3-methylcrotonyl-CoA carboxylase, geranyl-CoA carboxylase, mixed function oxidase, L-cysteine sulfonate decarboxylase and biotin transport;

thereby obtaining said first data set of differences from normal concentrations of metabolites, concentrations of enzymes, activities of enzymes or cellular functions.

13. The method of claim 1 wherein said disease or condition is selected from the

group consisting of a wound, cancer, multiple sclerosis, Alzheimer's disease, Parkinson's disease, depression, atherosclerosis, cystic fibrosis, diabetes, obesity, Crohn's disease and altered circadian rhythmicity.

16. The method of claim 13 wherein said disease is multiple sclerosis and said metabolites or enzyme activities comprise one or more of methyl transferase activity and S-adenosyl homocysteine.

18. The method of claim 13 wherein said disease is Parkinson's disease and said metabolites or activities comprise one or more of polyamines, nonspecific N-methylase, acetyl-L-carnitine, Ca.sup.2+ /calmodulin-dependent protein kinase II, lysolecithin, sphingomyelin, SAM and vitamin B.sub.12.

☐ 18. Document ID: US 6020139 A

L10: Entry 18 of 19 File: USPT Feb 1, 2000

DOCUMENT-IDENTIFIER: US 6020139 A
TITLE: S-adenosyl methionine regulation of metabolic pathways and its use in diagnosis and therapy

CLAIMS:

3. A method according to claim 1 or 2 wherein said disease or condition is selected from the group consisting of a wound, cancer, multiple sclerosis, Alzheimer's disease, Parkinson's disease, depression, atherosclerosis, cystic fibrosis, diabetes, obesity, Crohn's disease and altered circadian rhythmicity.

6. A method according to claim 3, wherein said disease is multiple sclerosis and said metabolites or enzyme activities comprise one or more of methyl transferase activity and S-adenosyl homocysteine.

8. A method according to claim 3, wherein said disease is Parkinson's disease and said metabolites or activities comprise one or more of polyamines, nonspecific N-methylase, acetyl-L-carnitine, Ca.sup.2+ /calmodulin-dependent protein kinase II, lysolecithin, sphingomyelin, SAM and vitamin B.sub.12.

13. A method according to claim 1 or 2, wherein said identifying step (b) comprises determining in said biological sample:

(1) the concentration of metabolites selected from the group consisting of:

(A) for Pathway 1, S-adenosylinethionine (SAM), S-adenosyl homocysteine (SAH), adenosine, homocysteine, glutathione, glutathione disulfide, cystathionine, .alpha.-.alpha.-ketobutyrate, cysteine, cystine, taurine, choline, betaine, dimethylglycine, methylglycine, glycine, serine, folate, tetrahydrofolate, methylene tetrahydrofolate, methyltetrahydrofolate, adenosine triphosphate, methylated vitamin B12, an O- or N-methylated product of a methylating enzyme and a demethylateci product of a demethylating enzyme,

(B) for Pathway 2, SAM decarboxylated adenosylmethionine, methylthioadenosine,

ornithine, putrescine, spermine, spermidine, N.sup.1 -acetylspermidine, and N.sup.1 -acetylspermine; and

(C) for Pathway 8, biotin, biocytin, and biotin-protein conjugates,

(2) the concentration or activity of enzymes, or the level of cellular functions, selected from the group consisting of:

(A) for Pathway 1, adenosylinethionine synthetase, N.sup.5 -methyltetrahydrofolate:homocysteine methyltransferase, betaine:homocysteine methyltransferase, cystathionine .beta.-syntdase, .gamma.-cystathionase, methylene tetrahydrofolate reductase, SAH hydrolase, an O-methyltransferase, an N-methyltransferase, adenosine deaminase, a glutathione synthetic enzyme and a glutathione degradative enzyme;

(B) for Pathway 2, adenosinemethionine decarboxylase, spermidine synthetase, spermine synthetase, spermidine-spermine N.sup.1 -acetyltransferase, polyamine oxidase, methylthioadenosine phosphorylase, ornithine decarboxylase and polyamine transport; and

(C) for Pathway 8, biotinase, pyruvate carboxylase, acetyl-CoA carboxylase, propionyl-CoA carboxylase, 3-methylcrotonyl-CoA carboxylase, geranyl-CoA carboxylase, mixed function oxidase, L-cysteine sulfonate decarboxylase and biotin transport,

thereby obtaining said first data set of differences from normal concentrations of netabolites, concentrations of enzymes, activities of enzymes or cellular functions.

☐ 19. Document ID: US 5856095 A

L10: Entry 19 of 19 File: USPT Jan 5, 1999

DOCUMENT-IDENTIFIER: US 5856095 A
**** See image for Certificate of Correction ****
TITLE: Identification of two novel mutant alleles of human thiopurine S-methyltransferase, and diagnostic uses thereof

CLAIMS:

1. An isolated polynucleotide molecule comprising a mutant allele of thiopurine S-methyltransferase (TPMT) or a fragment thereof, which is at least ten consecutive bases long and contains a point mutation at cDNA position 238.

2. An isolated polynucleotide molecule as claimed in claim 1, wherein said point mutation is a cytosine substitution for guanine.

4. An isolated polynucleotide molecule comprising a mutant allele of thiopurine S-methyltransferase (TPMT) or a fragment thereof, which is at least ten consecutive bases long and contains a point mutation at cDNA position 460.

5. An isolated polynucleotide molecule as claimed in claim 4, wherein said point mutation is an adenine substitution for guanine.

7. An isolated polynucleotide molecule comprising a mutant allele of thiopurine S-methyltransferase (TPMT) or a fragment thereof, which is at least ten consecutive bases long and contains a point mutation at cDNA position 719.

8. An isolated polynucleotide molecule as claimed in claim 7, wherein said point mutation is a guanine substitution for adenine.

10. An isolated polynucleotide molecule comprising a mutant allele of thiopurine S-methyltransferase (TPMT) or a fragment thereof, which is at least 260 consecutive bases long and contains a point mutation at cDNA position 460 and a point mutation at cDNA position 719.

11. An isolated polynucleotide molecule as claimed in claim 10, wherein the point mutation at position 460 is an adenine substitution for guanine and the point mutation at position 719 is a guanine substitution for adenine.

13. An isolated polynucleotide molecule fully complementary to any one of the polynucleotide molecules identified as SEQ ID NOS:1, 3, 5, or 7 or a fragment thereof, wherein said fragment is at least 10 bases long and contains at least one point mutation selected from the group consisting of G283C, G460A, and A719G.

14. A diagnostic assay for determining thiopurine S-methyl-transferase (TPMT) genotype of a subject which comprises

(a) isolating nucleic acid from said subject;

(b) amplifying a thiopurine S-methyltransferase (TPMT) PCR fragment from said nucleic acid, which includes at least one of cDNA positions 238, 460, or 719, thereby obtaining an amplified fragment; and

(c) sequencing the amplified fragment obtained in step (b), thereby determining the thiopurine S-methyltransferase (TPMT) genotype of said subject.

15. A diagnostic assay for determining thiopurine S-methyl-transferase (TPMT) genotype of a subject which comprises

(a) isolating nucleic acid from said subject;

(b) amplifying a thiopurine S-methyltransferase (TPMT) PCR fragment from said nucleic acid using a first and a second set of primers in a first and a second PCR reaction, respectively; wherein the first set of primers contains primer X and primer Y, and the second set of primers contains primer X and primer Z; wherein

(i) the Y primer is complementary to a region 5' to one of three point mutation sites at cDNA positions 238, 460, or 719, and includes the wild type nucleotide for said cDNA position;

(ii) the Z primer is identical to the Y primer except that instead of the wild type nucleotide, it contains the respective mutant nucleotide at the respective cDNA positions 238, 460, or 719; and

(iii) the X primer is complementary to a region 3' to the point mutation site corresponding to primers Y and Z;

(c) amplifying the sequence in between primers X and Y and in between primers X and Z; thereby obtaining an amplified fragment in each of the first and the second PCR reactions; and

(d) visualizing the contents of the first and the second PCR reactions, thereby determining the thiopurine S-methyltransferase (TPMT) genotype of said subject.

16. A diagnostic assay for determining thiopurine S-methyl-transferase (TPMT) genotype of a subject which comprises

(a) isolating nucleic acid from said subject;

(b) amplifying a thiopurine S-methyltransferase (TPMT) PCR fragment from said nucleic acid, which includes at least one of cDNA positions 238, 460, or 719, thereby obtaining an amplified fragment; and

(c) treating the amplified DNA fragment obtained in step (b) with

(i) CviRI in its corresponding restriction buffer to detect presence or absence of a point mutation at cDNA position 238,

(ii) MwoI in its corresponding restriction buffer to detect presence or absence of a point mutation at cDNA position 460, or

(iii) AccI in its corresponding restriction buffer to detect presence or absence of a point mutation at cDNA position 719,

thereby determining the thiopurine S-methyltransferase (TPMT) genotype of said subject.

18. A diagnostic assay for determining thiopurine S-methyl-transferase (TPMT) genotype of a subject which comprises

(a) isolating nucleic acid from said subject;

(b) making a first and a second PCR primer wherein

(i) the first PCR primer is complementary to a region 5' to one of three point mutation sites at cDNA positions 238, 460, or 719; and

(ii) the second PCR primer is complementary to a region 3' to the same one of the three point mutation sites at cDNA positions 238, 460, or 719;

(c) amplifying the sequence in between the first and the second primers; thereby obtaining an amplified fragment; and

(d) treating the amplified fragment obtained in step (c) with

(i) CviRI in its corresponding restriction buffer to detect presence or absence of a point mutation at cDNA position 238,

(ii) MwoI in its corresponding restriction buffer to detect presence or absence of a point mutation at cDNA position 460, or

(iii) AccI in its corresponding restriction buffer to detect presence or absence of a point mutation at cDNA position 719,

thereby determining the thiopurine S-methyltransferase (TPMT) genotype of said subject.

19. A diagnostic kit for determining thiopurine S-methyltransferase (TPMT) genotype of a subject comprising a carrier means having in close confinement therein at least two container means, wherein a first container means contains a first

polynucleotide molecule as claimed in claims 2, 5, 8, or 11, or a polynucleotide molecule complementary thereto and a second container means contains a second polynucleotide molecule encoding a wild-type allele of thiopurine S-methyltransferase (TPMT), a fragment thereof, or a polynucleotide molecule complementary thereto which is ten consecutive bases long and contains at least one of cDNA positions 238, 460, or 719, corresponding to the first polynucleotide of the first container means.

First Hit

L11: Entry 1 of 13 File: PGPB Jan 22, 2004

PGPUB-DOCUMENT-NUMBER: 20040014083
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040014083 A1

TITLE: Detection of heteroduplex polynucleotides using mutant nucleic acid repair enzymes with attenuated catalytic activity

PUBLICATION-DATE: January 22, 2004

INVENTOR-INFORMATION:

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DATE FILED: February 24, 2003

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INT-CL: [07] C12 Q 1/68

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REPRESENTATIVE-FIGURES: NONE

ABSTRACT:

Methods for detecting, localizing and removing abnormal base-pairing in a nucleic acid duplex are provided. These methods can be used for prognosis and diagnosis of diseases, disorders, pathogenic infections and nucleic acid polymorphisms. Combinations, kits and articles of manufacture for use in these methods are also provided.

RELATED APPLICATIONS

[0001] This application is a continuation-in-part application of U.S. patent application Ser. No. 09/514,016, filed Feb. 25, 2000, now pending. This application is also related to U.S. application Ser. No. 09/347,878, filed Jul. 6, 1999, entitled "COMPOSITIONS AND METHODS FOR ASSAYING ANALYTES" and U.S. application Ser. No. 09/457,205, filed Dec. 6, 1999, entitled "COMPOSITIONS AND METHODS FOR ASSAYING ANALYTES." U.S. application Ser. No. 09/457,205 is a continuation-in-part application of U.S. patent application Ser. No. 09/347,878, filed Jul. 6, 1999, now U.S. Pat. No. 6,376,210 B 1. The contents of each of these applications is incorporated herein in its entirety.